Déjà vu: coronary artery disease in monozygotic twins

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Short title: Coronary artery disease in monozygotic twins

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Coronary artery disease (CAD), the leading cause of death in Poland, originates in early life. [1] The lack of fully satisfying treatments strategies has inspired researchers to look at CAD from a different angle. Consequently, epigenetic factors have been postulated to play an important role in the early-life programming of adult health and diseases. [1] Due to the fact that twin pairs may experience different intrauterine environments, studies using phenotype-discordant monozygotic twins are particularly one of the most powerful study designs in epigenetic epidemiology. [1]

The largest prospective twin cohort study was conducted by Silventoinen et al. and was based on pooled twin data from Denmark, Finland, and Sweden. The authors acknowledged the existence of a clear aggregation of CAD mortality in twins, and especially in monozygotic twins (genetically identical). [2] Similarly, previous studies have shown concordance in symptoms of angina pectoris in twins. [2] A subsequent study by Hjelmborg et al. indicated that twinning studies seem to be vital to ensure the representativeness to the general population. [3]

The case reported in the current study provides an example of a monozygotic twin pair presenting with a similar lesion location in the left anterior descending artery (LAD) and an identical risk factor profile.

Twin A is a 53-year-old man. He was diagnosed with non-ST-segment elevation acute myocardial infarction and was treated successfully with the deployment of three sirolimus-eluting stents (CRE8 3.5 × 16 mm, CRE8 3.0 × 21 mm, and CRE8 2.75 × 16 mm) in the proximal and medial portions of the LAD [Fig. 1]. The patient was subsequently referred to our institute for further management. He did not have the coronary risk factors of obesity, diabetes mellitus, hypertension, or smoking; only dyslipidemia was present. Coronary angiography revealed a triple-vessel disease with subtotal (90%) occlusion in the proximal portion of I marginal and 80% discrete lesion in the distal portion of the left circumflex artery.
The patient was treated successfully with the deployment of two drug-eluting stents in the LCx/I marginal (bifurcation–CRE8 3.5 × 16 mm) and in the distal LCx (CRE8 3.0 × 16 mm) lesions. Transthoracic echocardiography revealed normal chamber diameters and a left ventricular function with a preserved ejection fraction (60%).

Twin B, the monozygotic twin brother of twin A, is a 53-year-old man. He was convinced to undergo a treadmill test, as he was experiencing dyspnea upon exertion. His only prevalent risk factor was also dyslipidemia. He had an electrocardiographically positive exertion test, so he underwent coronary angiography, which revealed a single-vessel disease with a 95% lesion in the seventh-segment LAD [Fig. 1]. The right coronary artery and LCx were free of disease. A sirolimus-eluting stent was successfully implanted later in the LAD. Transthoracic echocardiography revealed normal chamber diameters with hypokinesis of the anterior wall and a preserved ejection fraction (50%).

Little is known about the angiographic expression of CAD in twin pairs. Although previous observational studies have claimed that the location of coronary lesions is not strictly hereditary [4], the similar location of stenosis in the LAD, the age at first cardiac event, and the risk factor profiles show concordance in this pair of identical twins.

In conclusion, we suggest that when one twin presents with CAD, the second twin should be subjected to increased medical surveillance.
References


Figure 1

Left anterior descending arteries of the twin pair. Twin A–images below, Twin B–images above.

Twin B

Twin A